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Selective inhibition of PKC-β2 attenuates nitroglycerine-induced tolerance and prevents TNF-α induced toxicity in endothelial cells

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Introduction: Continuous treatment with organic nitrates causes tolerance and endothelial dysfunction by inducing reactive oxygen species (ROS) production, which is involved in PKCβ isoform activation. In the present study, we determine whether selective inhibition of PKC-β2 attenuates nitrate tolerance induced by chronic administration with nitroglycerine and inhibits TNF-α induced toxicity in endothelial cells.

Methods: Primary cultured human umbilical vein endothelial cells (HUVECs) were either not treated or treated with TNF-α (40 ng/mL) alone, or with TNF-α in the presence of CGP53353 (1 μM)), nitroglycerine (10μM), or CGP53353 plus nitroglycerine, respectively, for 24 h.

Results: TNF-α increased the levels of superoxide, NOx, MDA and nitrotyrosine production, accompanied by increased protein expression of p-PKC-β2 and endothelial cell apoptosis, whereas all these changes were further enhanced by nitroglycerine, indicating nitrate tolerance. Inhibition of PKC-β2 with CGP53353 decreased the protein expression of p-PKC-β2, and reduced TNF-α induced oxidative stress and cell toxicity with or without nitroglycerine.

Conclusions: It is concluded that selective inhibition of PKC-β2 attenuates nitroglycerine-induced tolerance and TNF-α induced toxicity in endothelial cells.

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